SUBCLAVIAN STEAL SYNDROME

A Review

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Abstract: The Subclavian Steal Syndrome is a condition that results from the stenosis or occlusion of the subclavian artery proximal to the origin of the vertebral artery. The blockage causes the reversal of the normal direction of blood flow in the vertebral artery which is termed the "steal", because it steals blood from the cerebral circulation. Blood is drawn from the contralateral vertebral, basilar or carotid artery regions into the low-pressure ipsilateral upper limb vessels. Although a relatively uncommon disease, it represents a condition which mimics many conditions often treated by chiropractors and osteopaths. The term subclavian steal syndrome is applied when reversed vertebral artery flow causes cerebral ischaemia with associated symptoms of vertebrobasilar hypoperfusion and/or symptoms of brainstem or arm ischaemia. This syndrome exists as an important consideration in the differential diagnosis of cerebral and brachial ischaemia.

Key Indexing Terns: Chiropractic, osteopathy, dizziness, vertebrobasilar ischaemia, subclavian steal, artery.

INTRODUCTION

Subclavian steal syndrome is characterised by attacks of transient cerebral ischaemic symptoms, which are often precipitated by exercise or work of the involved upper extremity. These symptoms are caused by the reversal of normal blood flow in the vertebral artery. This occurs in the presence of a stenotic or occlusive lesion of the subclavian artery proximal to the origin of the vertebral artery which is termed "steal", because it steals blood from the cerebral circulation. Hence blood is `stolen' from the basilar, contralateral vertebral or carotid arteries and redistributed into the low pressure ipsilateral vertebral artery and upper limb vessels. The term subclavian steal syndrome is applied when reversed vertebral artery flow causes cerebral ischaemia with associated vertebro-basilar and arm ischaemia symptoms (see tables 1 & 2). Patients who suffer from the subclavian steal syndrome present mostly as asymptomatic, and it is now recognised that a prerequisite for the development of symptoms is the presence of disease in other extracranial vessels supplying the brain (1-5).

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Table 1: Signs and symptoms of carotid artery iscahemia (39)
Hemianaesthesia
Hemiparesis or monoparesis
Headache
Dysphagia
Visual field disturbances
Confusion
Note: not all symptoms need necessarily be present

Table 2: Signs and symptoms of vertebrobasilar ischaemia (39)
Dizzyness / vertigo / giddyness / light head
drop attacks / loss of consciousness
diplopia
dysarthria
dysphagia
ataxia
nausea
numbness
nystagmus
Note: dizzyness is the most common, and is often unaccompanied by the other signs and symptoms

Although the subclavian steal syndrome is considered to be relatively uncommon, the incidence of this syndrome is thought to be more common than is recognised and diagnosed. Subclavian steal syndrome is slightly more prevalent in men than women, with the average age of patients being 61 and 59 years, respectively (1, 6).

DEFINITION AND HISTORICAL PERSPECTIVE

The subclavian steal is the result of stenosis or occlusion of the subclavian artery proximal to the origin of the vertebral artery causing reversal of the normal direction of blood flow in the vertebral artery. Blood is drawn from the contralateral vertebral, basilar or carotid artery regions into the low-pressure ipsilateral upper limb vessels. It is a relatively uncommon disease, reported to occur in approximately 6% of patients with asymptomatic cervical bruits (7). The term subclavian steal syndrome is applied when reversed vertebral artery flow causes cerebral ischaemia with associated symptoms of vertebrobasilar hypoperfusion and/or symptoms of arm ischaemia.

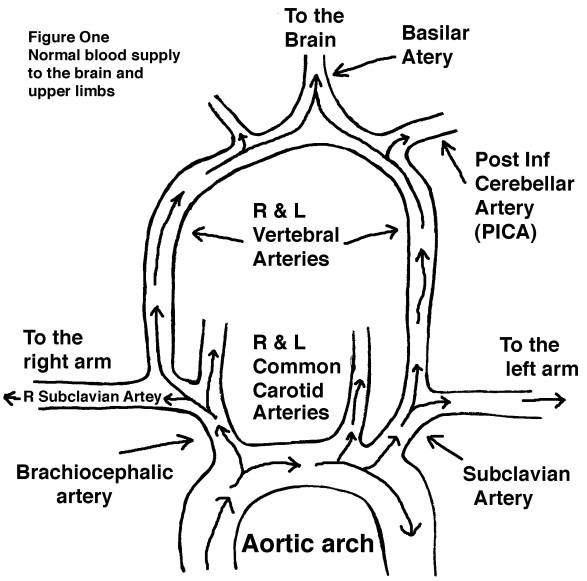
Occlusive disease of the subclavian artery has been recognised for more than a century. Contoni reports that in 1829 Harrison realised the importance of the vertebro-vertebral circulation in occlusions of the first portion of the subclavian artery (8). More recently Contorni (1960) used angiography to demonstrate retrograde flow in the vertebral artery ipsilateral to a proximal subclavian stenosis in a neurologically asymptomatic patient (8). In 1961 Reviech et al. reported cases similar to Contorni in patients with symptoms of cerebral ischaemia due to a decrease in pressure distal to stenosis below the level of the vertebro-basilar junction (9). The pressure gradient in the vertebral artery was decreased, thereby reversing cerebral blood flow. The pathologic pattern emerging from these findings was termed 'subclavian steal syndrome' by Fisher (1961) and has since been reported in a number of patients (10).

The largest study to date that has investigated subclavian steal was the Joint Study of Extracranial Arterial Occlusion. This was a cooperative study formulated in 1959 to determine the efficacy of arterial reconstructive surgery in the treatment of cerebrovascular disease resulting from surgically accessible lesions in the arteries of the neck and upper portion of the thorax (11). The study revealed that subclavian steal syndrome overall is slightly more prevalent in men than women. This is generally accepted despite some studies utilising more females for their research (12). The average age of sufferers was found to be 61 years in males and 59 years in females (12, 13). The average age is so high because of the rarity of atherosclerotic plaques being detected in the subclavian artery and its branches and the lack of patients presenting with symptoms.

ANATOMY AND PATHOGENESIS

The brain is supplied through an extensive system of branches from two pairs of vessels, the internal carotid arteries and the vertebral arteries (14). Branching from the common carotid arteries, the internal carotids supply 80% of the blood flow to the brain. Within the cranial vault they in turn branch into the middle cerebral artery and the anterior cerebral artery. The other 20% of blood flow to the brain is supplied by the vertebral arteries. They arise from the subclavian artery on the left and the innominate artery on the right. The right and left vertebral arteries unite at the caudal border of the pons to form the basilar artery which then branches into the two posterior cerebral arteries. The cerebral arteries join their communicating branches at the circle of Willis at the base of the brain which forms a complete vascular ring that encircles the diencephalon connecting the vertebro-basilar and internal carotid systems (see figure 1) (15).

Figure One
Normal blood supply to the brain and upper limbs

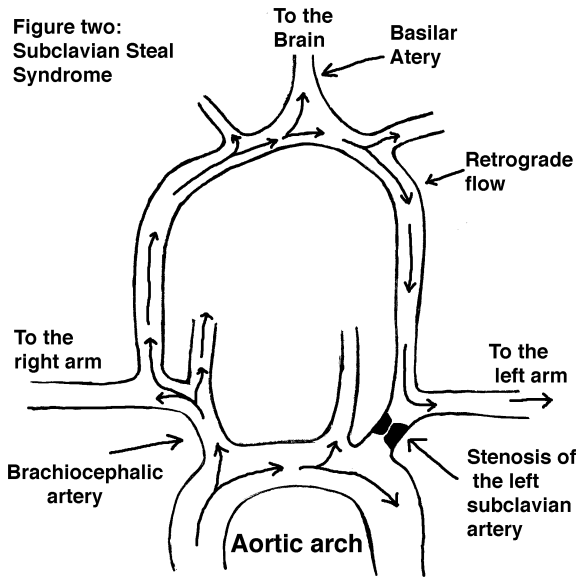


The purpose of the circle of Willis is to ensure maintenance of the circulation if one of the four main vessels is interrupted. Stenosis or occlusion of one of the subclavian or the innominate arteries, in the case of subclavian steal, results in an heightened pressure gradient in the affected vessel and a decreased pressure gradient in the ipsilateral vertebral artery. So there is an attempt to reconstitute distal arm flow by reversing the flow through the vertebral artery shunting flow away from the brainstem. This change in flow causes a reversal of blood flow in the vertebral artery resulting in the decreased posterior cerebral blood flow leading to symptoms of cerebral ischaemia.

Atherosclerosis is the major causes of arterial occlusive disease involving the subclavian artery. It is a degenerative disease of large and medium sized arteries, characterised by lipid deposition and fibrosis. Patients with atherosclerosis may be seen with either occlusive or atheroembolic symptoms. Smoking is a risk factor in 78% to 100% of cases and concomitant coronary artery disease is present in 27% to 65% of cases (15). The most common location for atherosclerotic lesions causing reversal of blood flow is the proximal portion of the left subclavian artery. Zimmerman reported a preponderance of 3:1 of symptomatic subclavian artery lesions on the left to the right (16). The right subclavian and innominate arteries are less common locations of atherosclerotic lesions that may result in subclavian steal in the proximal portion of the subclavian steal. Lesions of subclavian arteries distal to the vertebral artery origin are equally distributed between sides (16). The reason for this has not been postulated at this time.

Reversal of blood flow in the ipsilateral vertebral artery results from arterial occlusive disease in the subclavian artery. Consequently, blood is drawn from the

contralateral vertebral artery to the homolateral vertebral artery. With a left subclavian occlusion, maintenance of blood flow to the arm may occur because of reversal of flow from the basilar artery via the left vertebral. This flow from the basilar artery in the brain, which has received its circulation from the right vertebral artery or carotid artery via the circle of Willis, forms the basis of the subclavian steal syndrome (see figure 2).



SIGNS AND SYMPTOMS

Persons with atherosclerotic disease of the subclavian artery often do not have symptoms. It was originally believed that the majority of patients suffering from subclavian steal were symptomatic however Ehrenfeld et al (1969) reported that most cases were in fact asymptomatic (17). Haemodynamic reversal in the vertebral artery rarely, if ever, results in permanent neurologic deficit and in fact may be completely asymptomatic (17). Both asymptomatic patients and patients with hemispheric symptoms may show the same vertebrobasilar flow alterations as patients with undisputable vertebrobasilar symptoms. In fact retrograde vertebral artery blood flow may not necessarily indicate a steal from the cerebral circulation at all. Reversed vertebral artery blood flow has been described during angiography even when there was no stenosis present in the subclavian artery (18).

Vertebral-basilar symptoms that may occur with subclavian steal syndrome are multiple, diverse and often obscure (Table 1). The reasons for the variations in presentation are likely to involve a number of factors. In patients with haemodynamically significant subclavian artery stenosis the presence of reversed ipsilateral vertebral artery blood flow or the presence or absence of a radiologic steal were not good determinants of either the presence or type of presenting symptoms. With a progressive decrease in the amount of blood flow available to the brain, when there was reduction in circulation to the anterior and posterior regions, there was a decreased incidence of steal (18). Thus, rather than the steal determining the symptoms, perhaps the presence and the location of the extracranial vascular stenosis determines both the symptoms and the presence of reversed flow in the vertebral artery. The only exception was found in 14 patients who had no other significant stenosis and had non-hemispheric symptoms. In this group the incidence of steal was 65% (19).

Many symptoms reported may be related to associated severe carotid artery disease which can provoke symptoms suggesting vertebrobasilar insufficiency. In the Joint Study of Extracranial Arterial Occlusion more than 80% of 168 patients reviewed had associated extracranial carotid obstructions (13), a finding also supported by other observers (20). In 1996 Lacey reported that symptomatic subclavian artery lesions were also associated with concomitant lesions of the contralateral vertebral artery or of one or both carotid arteries in 35% to 85% of patients (15).

This has been reinforced by studies showing the restoration of normal blood flow by means of open surgery or transluminal angioplasty did not always lead to symptom relief. It is now recognised that a prerequisite for the development of symptoms is the presence of disease in other extracranial vessels supplying the brain. Although abundant collateral pathways around the shoulder and the redistribution of blood flow permitted by the circle of Willis make it unlikely that occlusion of the arch vessels (common carotid, brachiocephalic or subclavian arteries) in the upper thorax are responsible for symptoms of cerebral ischaemia (17). Walker et al found the presence and nature of cerebral symptoms were best determined by the location of other anterior stenosis; reduced anterior circulation in patients with hemispheric symptoms and reduced posterior circulation in patients with nonhemispheric symptoms (18).

The symptoms of cerebral ischaemia resulting from subclavian steal syndrome were thought to be associated with, or exacerbated by arm exercise. In most reported series in the literature, augmentation of the posterior circulation steal with arm exercise seldom reproduces the symptoms of posterior circulation insufficiency (20). In a number of studies, Magaard et al. studied cerebral blood flow using xenon washout studies in patients with subclavian artery occlusive disease (21). They found that even with a subclavian artery stenosis and retrograde vertebral artery blood flow, cerebral blood flow increased during arm exercise. Arm exercise induced brainstem dysfunction was only demonstrated in patients with bilateral subclavian steal phenomena in studies conducted by Hennerici et al (22).

It has been postulated that only one major blood supply to the intracranial circulation is required to prevent cerebral ischaemia. However, finding a complete circle of Willis is a relative rarity. Miller-Fisher, cited in Lacey (15), reported a lack of adequate collateral channels in the anterior circulation in 44% and a small posterior communicating artery in 49% of consecutive autopsy dissections. Therefore a combination of extracranial vascular stenosis plus an incomplete intracranial network may result in reduced blood flow to either anterior or posterior cerebral regions.

Because of extensive collateral circulation from the vertebral artery and around the shoulder, symptoms of arm ischaemia seldom develop from proximal subclavian artery occlusive disease alone. Welling et al.(23) reported in a group of 77 patients with proximal subclavian artery occlusions, 88% of the patients complained of CNS symptoms and only 40% noted intermittent claudication of the arm. In contrast to more proximal subclavian arterial disease, intermittent claudication of the arm is the most frequently noted symptom with distal subclavian occlusions (16). Arm symptoms associated with subclavian steal syndrome include ischaemic rest pain, ulcers, muscle fatigue and digital necrosis.

Frank digital necrosis is more often caused by distally shed emboli than atherosclerotic disease of the proximal vascular tree (16). Embolisation of atherosclerotic debris may result in digital artery occlusion with small areas of tissue loss or even significant hand ischaemia occurring (18). Walker et al. also found that patients with symptoms of arm ischaemia usually had an anaemic variant causing reduced arm circulation, resulting in a greater difference in arm blood pressures.

Systems of classification of subclavian steal have been recognised based on the territory from which blood is stolen or on vertebral artery haemodynamics. Four types of steal have been described: vertebro-vertebral, carotid-basilar, external carotid-vertebral and carotid-subclavian (13). The last shunt can only occur on the right side with brachiocephalic occlusion proximal to the origin of the carotid artery (See figure 3). Branchereau et al (20) recently described three patterns of flow in the vertebral artery in the presence of ipsilateral subclavian stenosis or occlusion. Haemodynamic abnormalities consisted of reduced anterograde flow in the vertebral artery (stage I), reversal of the flow during reactive hyperaemia testing of the arm (stage II) and permanent retrograde flow (stage III). The three stages correlated with severity of disease in the subclavian artery and all patients classified within stage III had occlusion of the subclavian artery. There is however, no clear relationship between the vertebrobasilar flow alterations and the clinical symptoms.

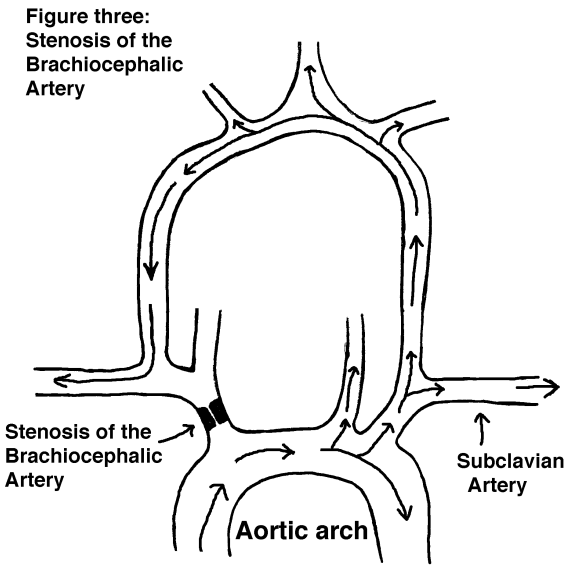


Figure three: Stenosis of the Brachiocephalic Artery

VARIATIONS TO THE SUBCLAVIAN-STEAL SYNDROME

Two important subsets of subclavian steal syndrome have been reported on in the literature. The coronary-subclavian steal syndrome is one subset and only occurs in patients who have undergone coronary artery revascularisation surgery utilising an internal bypass graft from the internal mammary artery(19, 21, 24). The aetiology, as in classical vertebral-subclavian steal syndrome is an occlusion or high grade stenosis in the proximal subclavian artery. The blood flow to the arm is then drawn from reverse flow in either, or both, the vertebral artery and internal mammary artery. It should be considered present if exceptionally prominent retrograde left internal mammary artery opacification or anterograde opacification of native coronary arteries or vein grafts is seen (21). In coronary-subclavian steal, the subclavian artery at the origin of the grafted internal mammary artery is either stenosed prior to the bypass procedure or becomes stenosed after bypass, allowing retrograde blood flow through the mammary artery (10, 25). Symptoms related to left upper limb or cardiac ischaemia may be present. Patients may also present with episodic left upper limb paraesthesias or claudication or symptoms related to myocardial ischaemia (19, 26). A bruit over the left subclavian region may also be present. Most cases of coronary-subclavian steal syndrome present within 3 years of surgery (25). This syndrome in patients, resulting in myocardial ischaemia, although rare, is becoming increasingly common with the increased use of the internal mammary artery coronary bypass grafts.

Another subset that has only recently been reported has been described as partial or pre-steal in physiology (27). The physiology of these partial steals has been described

as normal caudocephalad flow during diastole and reversed flow in the cephalocaudad direction during systole. However, lowered arm resistance is produced, when these patients are stressed with exercise and/or postocclusion hyperaemia and the vertebral flow becomes cephalocaudad in both systole and diastole (28, 29). The converted complete steal pattern in these patients will revert back to a partial to and fro pattern after a period of rest. The clinical significance of these partial subclavian steal lesions, however, like complete steals, is not completely certain at this time (27).

DIAGNOSIS

When subclavian steal is present in association with neurological symptoms or arm ischaemia, the diagnosis of subclavian steal syndrome can be made following the exclusion of other causes of these symptoms. The importance of the history and examination is paramount in the detection of the subclavian steal syndrome, and the key points of these investigations are presented in tables 3 & 4.

Table 3: Summary of key history questions
How, when, where the problem began
Where is / are the major complaints felt
Quality of the symptoms
Aggravating and relieving factors
Any radiation of pain
Past treatment for complaint

Table 4: Summary of key examinations
1. Chiropractor / Osteopath
History
The taking of blood pressure bilaterally
Auscultation of bruits
Arm exercise (swinging the arm around) in the attempt to reproduce symptoms
2. Medical
Doppler ultrasound

Measurements of the upper extremity blood pressure and pulse are routine procedures carried out during most clinical examinations. However, a bilateral assessment is required in the case of the subclavian steal. A discrepancy in the bilateral assessment of these vital signs is not always significant, however they may be of particular interest in association with vertebro-basilar symptoms or symptoms of arm ischaemia. Chiropractors and osteopaths are often the first practitioners to evaluate the older patient with subclavian steal syndrome, therefore awareness of the implications of these findings is paramount.

A blood pressure that is lower than expected for an older patient, and a weak or diminished radial pulse occurring ipsilaterally, should persuade the examiner to assess the vital signs in the contralateral arm. A difference in systolic blood pressure between arms is suggestive as being one of the signs of subclavian steal (12, 15). Patients who have symptoms of upper extremity arterial insufficiency such as claudication will most often have a contralateral brachial pressure difference of 40mm Hg to 50mm Hg. Those with predominantly cerebral symptoms usually have a differential between 20mm Hg and 40mm Hg, because of the reconstitution of the arm flow through the vertebral artery (16, 30). Bilateral arm pressure should also be measured in any patient with suspected diffuse vascular disease, particularly those with suspected cerebrovascular symptoms. Auscultation should also be performed for the evaluation of carotid and suprascapular bruits, which are often present in a subclavian steal. In patients with the above findings, a thorough history and physical examination should determine the presence of vertebro-basilar symptoms or symptoms of arm ischaemia described above. In some patients, their vertebro-basilar symptoms may be exacerbated by the brachial artery compression-decompression test. This test involves the arm being compressed with a blood pressure cuff above the systolic blood pressure level for 3 minutes. At decompression, there will be a sudden increase in the steal phenomenon in the vertebral artery due to reactive post-ischaemia arm hyperaemia (31). This generally results in the presence of the patient's vertebro-basilar symptoms, and is a good diagnostic tool in the partial diagnosis of a subclavian steal syndrome.

In the case of a suspected subclavian steal syndrome, a thorough clinical history and examination and the ruling out of all of the other sources of symptomatology is vital for diagnosis. To complete the diagnosis, other testing is often warranted. Of these tests, Doppler ultrasonography has been demonstrated to be the most sensitive and specific instrument in detecting subclavian steal syndrome (see table four) (13, 18, 22, 31). Apart from being safe and highly accurate, Doppler ultrasonography is non-invasive, such that it does not alter the hemodynamic patterns in subclavian steal and can be repeated as required without being detrimental to the patient. Doppler scanning also allows simultaneous imaging and flow mapping of the vertebral artery in addition to evaluation of the subclavian and carotid arteries. Accurate analysis of the status of the entire extracranial vascular tree is important to rule out other atherosclerotic lesions. Performing the reactive hyperaemia testing of the arm during Doppler examination may be useful to enhance the steal phenomenon and confirm reversed vertebral flow in patients where the diagnosis might otherwise have been uncertain. Insonation of the basilar artery and arteries of the circle of Willis by transcranial Doppler ultrasonography is now possible with complete characterisation of all possible flow patterns (13).

Lacey places more importance on contrast angiography in the diagnosis of subclavian steal syndrome (15). Lacey suggests that Doppler ultrasonography should be used as the initial screening instrument and if the ultrasonograph finds the presence of a subclavian lesion, a confirmatory contrast arteriogram is required. Doppler ultrasonography is believed by Lacey to be less accurate in diagnosing a subclavian steal syndrome when compared to angiography (15). This is because of its inability to fully view the anatomy as a result of bony structures such as the clavicle and first rib overlying the subclavian artery. However other authors have shown retrograde vertebral artery blood flow demonstrated during contrast angiography, to not necessarily be indicative of a subclavian steal from the cerebral circulation (22). This is because reversed vertebral artery blood flow has been described during contrast angiography even when a subclavian lesion was not present. The forceful injection of a large bolus of dye into a relatively small artery was considered the cause of this artificially reversed vertebral artery blood flow (18).

TREATMENT OF THE SUBCLAVIAN SYNDROME

Management of the subclavian steal syndrome has traditionally been treated surgically (13, 32, 33). However, recent trends have leaned towards percutaneous transluminal angioplasty (PTA) or percutaneous transluminal coronary angioplasty (PTCA) as a valuable alternative to surgery. Other methodologies which have generated some interest in recent times include artherectomy and stenting.

The aim of treatment is to restore permanent antegrade blood flow to the effected vertebral artery thus abolishing cerebral hypoperfusion and it's associated symptoms. Surgical procedures of choice are extra-thoracic extra-anatomic bypass. These include:

Carotid-subclavian bypass

This procedure is well standardised and consists of placing a short common carotid-subclavian artery interposition graft. Prosthetic grafts, such as Dacron (Du Pont, Stevenage), polytetrafluoroethylene or Teflon prosthetic vessel (13, 27, 34) have been shown to be far superior to saphenous vein graft in the same position. Clinical and experimental evaluation have over ridden concerns that such a bypass would create post-operative carotid steal. This type of steal was found only to occur if proximal common carotid stenosis was not corrected at the time of revascularisation. A review of 426 patients between 1962-80 showed a mortality rate of 2.4%, morbidity 5.4% and a five year graft patency rate of 94% (13, 34).

Carotid-subclavian transposition

The subclavian artery is divided close to it's origin in this procedure. The distal end is then anastomosed end to side to the common carotid artery. The advantage of carotid-subclavian transposition is in the use of only one anastomosis site without the need for any synthetic material. This procedure is indicated in the presence of distal embolisation from the subclavian artery lesion (13, 34).

A retrospective look between the carotid-subclavian bypass and transposition showed no significant difference between operation time, complication rate, morbidity or mortality. However the patency rate after ten years for transposition was 100% compared to that of bypass at 74%.

Extra-anatomic bypass with the contralateral subclavian or axillary artery as an inflow source (axilloaxillary bypass)

Axilloaxillary bypass is an alternative procedure to carotid-subclavian bypass or transposition in the presence of associated disease of the ipsilateral carotid artery where there is risk of subclavian steal. Axilloaxillary bypass has the advantage that it is technically simple and does not require the need for supraclavicular dissection or carotid clamping. Treatment of 237 patients over twelve years showed a mortality of 0.4% and graft occlusion in 10.5% of patients (13).

Carotid endarterectomy

In the presence of coexisting carotid artery disease, carotid endarterectomy should be considered. It is uncertain whether subclavian revascularisation without carotid endarterectomy eliminates vertebro-basiliar symptoms, however, retrospective studies show a combined approach gives the best chance of symptom relief in the presence of coexistent carotid artery disease and subclavian steal (13).

Percutaneous Transluminal Angioplasty

PTA is a relatively new procedure incorporated in the management of subclavian steal syndrome. PTA has been shown to successfully dilate stenosis and even occlusions at many sites, thus abolishing vertebrobasiliar symptoms associated with the subclavian steal syndrome. There are definite benefits in the use of PTA as it can be performed in a day case setting under local anaesthetic. Immediate complication rates of subclavian angioplasty is less than 10 percent and complications are to a far lesser degree to that of surgery. However, long term patency rates of PTA appear to be inferior to that of the fore mentioned procedures at a restenosis rate of 14% at 37 months. Further angioplasty may be used to dilate the affected site (13, 33).

PTA was introduced in 1980 according to Marques et al (33). In a literature review of over 400 cases since has shown an immediate success rate in the range of 88-100% (33). Those unsuccessful was mainly due to technical failure seen in occlusions or very tight stenosis. Complication rates with PTA averaged 5% (range 0 - 10%) and without mortality. The complications seen in PTA were to a lesser degree compared to that of surgery. Those commonly seen were related to that of the puncture site (eg. formation of haematoma, spasm of the brachial artery and thrombosis formation at the site) or at the site of dilation (eg. local dissection, formation of thrombosis or pseudoaneurysm). Severe subclavian artery dissections and restenosis can be managed with stent implantation (35). Primary stent placement in occlusive subclavian disease have to date seem promising (36). Vertebral embolic complications in PTA is around 1%. On average, a re-stenosis rate of 13% were seen at 30 months follow up (32, 33, 37).

Heparin is administered intravenously to patients prior to PTA. Lesions are crossed with a guidewire and balloon catheter via the femoral or brachial approach. Crowe & Iannone (1993) believe that assuming that the lesion is stenosed and not occluded, crossing the lesion should always be possible via the femoral approach (32). Even in occluded lesions have a good chance with this procedure and thus the brachial approach is rarely needed. Marques et al (33) believe the brachial approach gives better pushability and thus either or both approaches may be employed. The 3 - 4cm balloon catheter is inflated once to three times in a 1:1 ratio with the proximal subclavian artery diameter for 15 - 30 seconds.

There are some concerns that embolic material may flow to the cerebrovasculature during PTA and some authors suggest placement of a small balloon in the vertebral artery concerned during dilation. However, it has been shown that there is a delay (ranging from 20 seconds to 20 minutes) following successful subclavian PTA before antegrade flow is restored to the ipsilateral vertebral artery (26, 33). This suggests that embolic material is diverted to the extremities to protect the cerebrovasculature from invasion.

Residual stenosis immediately following PTA should be no greater that 30% and with a transstenotic pressure gradient or bilateral upper limb blood pressure reduced to near 0mmHg. Simple systemic review and physical examination should pick up recurrences. Follow up doppler examinations should be performed every 6 months and repeat catheterisation should be performed in patients who become symptomatic or present with an upper limb blood pressure difference of greater than 20mm Hg.

Artherectomy

Artherectomy of the subclavian artery for the treatment of the coronary-subclavian steal syndrome is a relatively new procedure with the first report case in 1993. Directional artherectomy consists of using a peripheral artherectomy catheter (Simpson Atherocath, Devices for Vascular Intervention) via a percutaneous femoral approach. Multiple cuts are made in the lesion with residual stenosis to be no greater than 30% (27).

Artherectomy of peripheral vasculature appears to have a slightly less rate of restenosis than that of coronary arteries. This probably stems from the simple fact that the lumenal diameter of peripheral arteries are greater than that of the smaller coronaries.

Directional artherectomy appears to be an effective and safe methodology in the management of subclavian steal syndrome, especially in the presence of concomitant carotid disease where surgical intervention would not be feasible. It also appears to hold a slight advantage over PTA where the incidence of severe dissection, abrupt subclavian closure and complication rates are lower. However long term follow up will be necessary to determine the outcome of artherectomy.

Stenting

Primary stenting of the subclavian artery in the treatment of the subclavian steal syndrome and may reduce the restenosis rate and thus improve the long term patency rates of treatment. Stent placement may also be useful in bridging dissections or after failed/insufficient PTA. In theory, stent placement may also decrease the rate of embolic material by trapping it against the artery wall (38).

The procedure is similar to that of PTA with the introduction of the stent inserted with the use of a guidewire, catheter and balloon. High success rates along with low complications have created much interest. Incidence of recurrence and long term outcomes will require follow up studies (35, 36).

Summary of treatment approaches

Treatment is aimed at restoring permanent antegrade blood flow to the affect vertebral artery and thus abolishing vertebral-basiliar symptoms and other manifestations of this disorder.

Traditionally, extra-thoracic extra-anatomic bypass surgery has been the modality of choice in the management of this disorder. These procedures are well established, but come with a significant risk of morbitity and mortality. With the introduction of PTA in 1980, more and more are advocating PTA as the conduit of choice. PTA is effective, can be performed in a day case setting under local anaesthetic and possesses far less as well as less significant

complications as opposed to surgery. Long term efficacy appears to be somewhat inferior to that of surgery. Re-dilation and additional techniques such as stenting and endartherectomy in conjuction with PTA have helped improved patency rates. Arterectomy has been the most recent addition in the management of the subclavian steal syndrome and intial results have been promising.

It is also important to not overlook dietary and lifestyle factors as the subclavian steal lesion is, on the whole, atherosclerotic. These factors must be addressed pre and post-treatment in order to gain maximal efficacy.

CONCLUSION

Subclavian steal syndrome is a relatively uncommon benign condition reported to occur in approximately 6% of patients with asymptomatic carotid and supraclavicular bruits (12). It has been well documented that most patients who are diagnosed as having a reversal of blood flow in the vertebral artery, are asymptomatic. The disabling symptoms that do occur are probably associated with inadequate collateral circulation or associated extracranial atherosclerotic disease. The left sided lesion in the subclavian artery, just proximal to the vertebral artery is seen to predominate, and the symptoms are generally neurogenic in nature, or involve upper extremity claudication, or both.

The most common presenting symptoms found in subclavian steal syndrome are vertigo, syncope and intermittent claudication of the ipsilateral upper extremity. In addition several patients suffer ischaemic hand symptoms from low blood flow rates. Visual disturbances and transient paralysis are most commonly found in patients with co-existent carotid disease, whilst patient's whose chief complaint was upper extremity claudication, did not have associated carotid stenosis (12).

Through performing thorough histories and physical examinations, a chiropractor may be the first person to identify a patient with subclavian steal syndrome. All patients initially seen with discrepant bilateral blood pressures or a diminished/weakened radial pulse unilaterally should have the diagnosis of subclavian steal considered during their evaluation. In addition patients with vertebro-basilar symptoms or arm ischaemia symptoms should have the diagnosis of subclavian steal ruled out. This can be achieved through the use of modern imaging techniques such as Doppler ultrasonography and angiography.

In cases of asymptomatic subclavian steal, it is important that the patient understands their diagnosis, and are made aware of the symptoms associated with the worsening of their condition. Patients also need to be reassured that most subclavian steal cases are benign, and that if their condition does progress, it is a treatable non-life threatening problem. Finally patients should also be educated about the atherosclerotic disease process and its associated risk factors.

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